

NOTES ON THE DISEASES OF TROUT AT THE MAHILI HATCHERY—KULU (PUNJAB).

BY

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(With a plate).

Little attention has so far been given to the diseases of trout in its Indian habitats. The control of parasitic and other diseases is a problem of considerable importance. Under domestication, i.e. when herded together and fed on artificial food, trout, like all other animals, fall victims to parasites and contract diseases.

Gaschott (1931) has given an account of the various diseases, to which trout are susceptible, and has dealt with their diagnosis and treatment. Davis (1936) has dealt with internal and external parasites of trout and such other diseases as Fungus, Pop-eye, Thyroid tumour, Intestinal inflammation, White spot disease, Blue sac disease and Soft egg disease. Moore (1923, 1924) has made a study of diseases of fish in State Hatcheries in New York. Carcinoma of Thyroid in the Salmonoid Fish has received an exhaustive treatment in the well illustrated paper by Gaylord and Marsh (1914). Several other investigators, both in Europe and America, have made a study of the various parasites and diseases of trout and other fish and dealt with their control and treatment.

A systematic study of the diseases of trout, it is regretted, has not been made on account of lack of facilities, and in the present paper an account of such diseases as have come to our notice at the Punjab Government Trout Hatchery at Mahili (Kulu) is presented.

The diseased trout were obtained from the Hatchery and most of this material was received preserved in 5 per cent formalin. In a few cases alive sick fish were also examined. Of the two species, Rainbow Trout and Brown Trout cultivated at the Mahili Hatchery, the former has suffered far more acutely from parasites and other diseases than the latter.

The commoner diseases at the Mahili Hatchery have been Fin-rot, Carcinoma of Thyroid, Inflammation of Intestine, and Fungus.

Fin-rot or Tail-rot or Fin-disease was first noticed at the Hatchery in August 1929 among Rainbow Trout, and it appeared in an epidemic form. The diseased fish had grown sluggish and dark in colour. The dying fish came to the surface, gasped for air, turned upside down and after a few hours died. Both in the adult and in the fingerlings the caudal fin had lost its rays and had been reduced to a stump (Fig. 1). In most of the cases dorsal,

pectoral and anal fins had also been affected and showed naked fin rays without any covering of epidermis.¹

Fin-rot is caused by a rod-shaped bacterium which can usually be found in large numbers in the infected fins (Davis 1936). The infection usually starts from the outer margin of the fin, where the epidermis becomes thickened and forms a white line, visible externally across the fin. The fin rays are also attacked and soon become frayed and broken. Sometimes only a portion of the fin is destroyed, and in other cases the outer half or two-thirds is destroyed.

The young fingerlings usually die before the infection has spread to the underlying tissues; while in the adults the tissues at the base of the fins are also affected. The disease varies greatly in its intensity. Among the fry the mortality is always heavy. Among adults recovery is possible though the fins retain their stumpy appearance. According to Davis (1936) 'the fins regenerate more or less completely'. No such regeneration of the fins, however, has so far been noticed among the fish which have recovered at the Mahili Hatchery.

According to Davis (1936) 'little information has been obtained regarding the factors that tend to bring an outbreak of the disease'. At the Mahili Hatchery, however, it has been definitely observed that fish and fry reared in clear spring water remain unaffected; while those reared in ponds fed by river water, which brings in silt during the floods, always suffer from an attack of Fin-rot soon after the monsoon rains. At Madhopur Farm, too, flood water in July was directly responsible for the outbreak of this disease among Rainbow Trout fry.

Control.—A salt bath instead of producing any healing effect, seemed to hasten the death of the sick fish at the hatchery. Davis (1936) recommends baths of a solution of copper sulphate, 1 : 2,000, for one or two minutes. But it is too strong a solution to be administered by unskilled hands either to adult fish or to fingerlings. A bath of 1 : 20,000 solution of copper sulphate for 10 to 15 minutes however, proved efficacious in early stages of the disease. The bath was repeated daily and healed the fins within a fortnight. The fish in which the disease had advanced did not survive. The warm water bath, recommended by Taplin (1932), proved beneficial during winter. The fish from the hatchery pond, where the range of temperature was 38°F. to 48°F., were transferred to a spring fed pond with a temperature ranging from 55°F. to 58°F. and were cured of the disease completely.

Carcinoma, Thyroid tumour or Goitre in trout is said to be analogous to goitre in man (Davis 1936). It is, therefore, interesting to note that in the Kangra District, where Mahili Trout Hatchery is located, human beings, too, suffer from goitre.

¹ Dr. W. Rushton (Fishmongers Hall, London), to whom the diseased Rainbow Trout were sent for examination, reported that 'No external lesions could be found or any features, which point to, or suggest, that the fish had died from "furunculosis" . . .'

Note.—Furunculosis is another bacterial disease, the most characteristic symptom of which is the presence of open sores on the body (Davis 1936).

The disease has not so far been noticed in an epidemic form at the Hatchery. Only occasionally one or two fish have suffered. The tumours of thyroid, in the diseased fish examined (Figs. 2 and 3), were located at the junction of the first and second pair of gill arches, on either side, and also between the third and the fourth gill arches. The former pair of tumours were visible internally too, on the floor of the mouth. Atrophied gill filaments were seen attached to the tumours.

The primary cause of thyroid tumour is now generally considered to be a deficiency of iodine, which is essential to the proper functioning of the thyroid gland (Davis 1936). Overcrowding, a limited supply of water, and insanitary condition in the ponds also appear to be important contributing factors.

The diseased fish linger on for months and months, and it is only when the tumour has enlarged to such an extent that it has spread into the gill arches, that the blood vessels cease to function and in portions the circulation is stopped and the fish dies.

Control.—At the Mahili Hatchery the fish in which the disease was noticed in an advanced stage were always killed. In the early stages of the disease a weak bath of iodine (potassium iodide) in 1 : 1,000,000 strength has given satisfactory results, and the fish recovered. Gaylord and Marsh (1914) recommend the administration of the following chemical baths: iodine (potassium iodide) 1 : 1,000,000; arsenic (arsenic oxide) 1 : 3,000,000, and mercury (mercuric chloride) 1 : 5,000,000. Davis recommends (1936) addition of iodine directly to the food: a table-spoonful of 1 per cent iodine dissolved in 1 per cent solution of potassium iodide 'thoroughly mixed with about 50 pounds of ground food is sufficient to keep the fish from showing any trace of thyroid tumour'.

Intestinal Inflammation: Most of the Rainbow Trout attacked by Fin-rot at the Hatchery suffered from inflammation of the intestine as well. In 1929 when the epidemic of Fin-rot appeared, the fish, as an economical measure, were being fed on 75 per cent oat flour and 25 per cent dried fish. It was rather expecting too much from these carnivorous fish to maintain their vitality on such a poor diet. The diseased fish had grown dark in colour, refused to take any food and sought the corners of the pond. The intestine was inflamed and contained only mucus. One Brown Trout that died in February 1930 had a large tumour in the intestine which filled up the entire lumen.

The disease is apparently caused by unsuitable food. Change of diet, recourse to natural food, such as aquatic insect larvae, worms, snails, slugs and a large proportion of meat in their diet improves the condition of the fish.

Fungus or *Saprolegnia* has so far not caused any serious damage at the Hatchery. *Saprolegnia* readily attacks dead eggs, dead fry or any dead organic matter lying in the rearing or hatching boxes. Adults are attacked by the fungus when they have been handled roughly, especially after the stripping season. It appears as tuft of white threads—mycelial filaments—on any part of the fish after

some physical injury, and its filaments soon spread into the underlying tissues.

Control.—A 3 per cent solution of common salt for 5 to 10 minutes or until fish show signs of distress, has so far proved very efficacious. Patches of skin overgrown with fungus, when rubbed gently with cotton soaked in a 1 : 2,000 copper sulphate solution or solution of common salt in vinegar or iodine, have effected speedy recovery. The most effective remedy to prevent an attack of fungus is to remove the root cause of the disease. Daily picking of dead eggs, cleaning all the troughs and hatching trays, regular salt baths for the fish, after they have been handled, have always proved effective against an attack of fungus.

The writer had an experience of a very striking example of *Saprolegnia* growth on Carp. In a Carp Farm in the Punjab the fish were confined to a portion of tank with a wire netting screen as a partition. Whenever fresh water was let into the tank, the fish rubbed their heads against the wire netting attempting to escape towards the inflowing current. They consequently received injuries on their lips, mouth and head, where fungus appeared in profusion and the fish began to die in large numbers. The wire netting screen was at once removed, the diseased fish were given salt baths and in a very short time recovered.

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